had been advised by certain attorneys that it was undesirable legislation."

Nothing was done thereafter until this year—1925. Mr. Oscar Mueller, representing the Los Angeles Bar Association, had been with me most actively in all our efforts to get this bill enacted into law. Mr. A. H. Koebig, representing the Society of Engineers; Dr. Thomas J. Orbison and Judge Frank Oster had also given sympathetic and valuable assistance.

In 1914, Mr. Mueller and I, at our own expense, visited various cities of the state and made addresses before bar and medical associations in behalf of this legislation. On invitation, we addressed a joint meeting of the San Francisco Bar and Medical Association. From time to time we made reports as chairmen of our several committees to our local and state associations. This year we determined to make one final effort to secure the passage and executive approval of the bill. Senator Jones of San Jose, always a loyal and indefatigable worker for the bill, did the most to make possible its passage in the Senate. At one time it was tabled in the Judiciary Committee of the House and seemed destined to fail of passage, but it was finally taken up and put through. Letters favoring its passage were written by university presidents and justices of the Supreme, Appellate, and Superior Courts, and sent to Sacramento. When Governor Richardson signed this bill he was kind enough to say publicly, "he considered it one of the best enactments of this Legislature."

What will be the effect of the statute in facilitating and dignifying the giving of expert testimony in the courts of California?

1. It will greatly reduce the number of experts called in a case and have thus a distinct economic value.
2. It will facilitate and expedite the trial of a case when the court calls its own expert.
3. It will dignify expert evidence, for the court's expert is an officer of the court and free from prejudice for or against plaintiff or defendant.
4. It does not invade the right or privilege of plaintiff and defendant calling their own experts.
5. It secures the expert of the court at a reasonable and just compensation, shared by the litigants and the state.

Finally, for eight or ten years Superior Judges of this city have, with consent of opposing counsel, called the court's expert with the greatest satisfaction to all and to very great economic advantage. So that long before the Act passed into law, the courts here had abundantly proved its practical value by showing its reasonable and just merits in the trial of their civil and criminal cases.

Recently, in the criminal case of the People vs. Young, the court called two alienists to pass upon the sanity of the defendant. The result of this first instance of record where the statute providing for the court's expert has been put into effect will be awaited with much interest.

Merritt Building.

THE NEW LAW

An Act to amend the Code of Civil Procedure of California by adding thereto a new section to be numbered and known as Section 1871, relating to experts, their appointment by the court, or a judge thereof, and providing for their compensation and manner of examination as witnesses.

The people of the State of California do enact as follows:

Section 1. A new section is hereby added to the Code of Civil Procedure of California, to be numbered and known as Section 1871, and to read as follows:

——. Whenever it shall be made to appear to any court or judge thereof, either before or during the trial of any action or proceeding, civil or criminal, pending before such court, that expert evidence is, or will be required by the court or any party to such action or proceeding, such court or judge may, on motion of any party, or on motion of such court or judge, appoint one or more experts to investigate and testify at the trial of such action or proceeding relative to the matter or matters as to which such expert evidence is, or will be required, and such court or judge may fix the compensation of such expert or experts for such services, if any, as such expert or experts may have rendered, in addition to his or their services as a witness or witnesses, at such amount or amounts as to the court or judge may seem reasonable. In all criminal actions and proceedings, such compensation so fixed shall be a charge against the county in which such action or proceeding is pending and shall be paid out of the treasury of such county on order of the court or judge. In all civil actions and proceedings such compensation shall, in the first instance, be apportioned and charged to the several parties in such proportion as the court or judge may determine and may thereafter be taxed and allowed in like manner as other costs. Nothing contained in this section shall be deemed or construed so as to prevent any party to any action or proceeding from producing other expert evidence as to such matter or matters; but where other expert witnesses are called by a party to an action or proceeding they shall be entitled to the ordinary witness fees only, and such witness fees shall be taxed and allowed in like manner as other witness fees. Any expert, so appointed by the court, may be called and examined as a witness by any party to such an action or proceeding or by the court itself, but when called shall be subject to examination and objection as to his competency and qualification as an expert witness and as to his bias.

Such expert, though called and examined by the court, may be cross-examined by the several parties to the action or proceeding in such order as the court may direct. When such witness is called and examined by the court, the several parties shall have the same right to object to the questions asked and the evidence adduced as though such witness were called and examined by an adverse party.

The court or judge may at any time before the trial or during the trial limit the number of expert witnesses to be called by any party.

THE PRE-OPERATIVE PREPARATION AND SURGICAL TREATMENT OF CHRONIC SPLENIC ANEMIA (BANTI'S DISEASE)

By Leo Pecci Bell, M. D., *Woodland, California*

Splenic anemia patients, in a well-advanced state, should be splenectomized as soon as possible after careful preparation by transfusion and rest. If splenectomy is resorted to before an advanced degree of cirrhosis has taken place, complete and permanent cures are obtained. Great care should be observed not to operate in a state of acute thrombo-phlebitis with temperature.

WHEN discussing the function of the spleen and its diseases, I am reminded of the story W. J. Mayo so frequently tells of the professor who asked a student to describe the function of the spleen. He stammered and stuttered and finally said that he had known but had forgotten. Whereupon the professor replied: "I am sorry you have for-

gotten because you appear to be the only one who has ever known."

It is definitely known that the spleen and liver of the foetus are blood producers; the liver up to about the fourth month, the spleen up to the fifth month. But, before birth, they lose the power of producing red blood and the spleen becomes a destroyer of debilitated red cells. The spleen begins to show senile changes at puberty, and after this time is more likely to show pathologic change.

Anatomically, the spleen is closely associated with the foregut. Its blood supply, which comes from the celiac axis, is altogether too large for its own nutrition. Moreover, when the arteries enter the spleen they lose their outer coats and become sinuses in which only the endothelium lies between the splenic pulp and the vessels. The fact that the splenic veins do not primarily enter the general circulation but become part of the portal circulation suggests that, whatever may be the action of the spleen on the blood, it is not completed and the splenic blood must pass through the liver to be acted upon there before it can enter the general circulation.

The sympathetic nervous system provides a close network about the splenic artery and its branches, and this in turn connects with the solar plexus and so with the adrenal glands. The vagus enervates the muscle fibers in the connective tissue. There has been some experimental work on the possible relationship between the spleen and the thyroid and thymus glands, but it is not conclusive.

Gaskill has proven that the spleen has very little anatomic nerve supply and deduces from this fact that it has no important internal secretion, since all organs of internal secretion have an abundant anatomic nerve supply. As proof of lack of internal secretion the normal spleen can be removed without any apparent disturbance.

Splenic anemia was first described by Gretsel in reporting a case from the clinic of Professor Greisinger, who had recognized lymphatic anemia and splenic anemia, and who used these names in his clinical dissertations to distinguish them from a large group of pseudo-leukemias, so-called. Banti, by collecting the pathological studies of Lodi, Concato, and Franzoline and the clinical studies of Greisinger and Gretsel, and by his clinical observations and necropsy studies in three cases, further separated this disease from a large group of splenomegalies. By his description of a disease which has been given his name, Banti stimulated investigation of disease of the spleen and particularly of splenic anemia.

Osler classifies splenic anemia as a primary disease of the spleen of unknown origin. It is characterized by progressive enlargement. Attacks of anemia are accompanied by a tendency toward hemorrhage, small in some cases, and by a secondary cirrhosis of the liver, with jaundice and ascites. That the spleen itself is the seat of the disease is shown by the fact that complete recovery follows its removal.

Rolliston, discussing this group described by Osler as "Primary Splenomegaly With Anemia" under separate headings, "Chronic Splenic Anemia" and "Banti's Disease," says he considers chronic splenic anemia as presenting the following characteristics: (1) Chronic splenomegaly, which cannot be correlated with any recognized cause; (2) the absence of enlargement of lymphatic glands; (3) chronic anemia with low color index; (4) the absence of leucocytosis and usually the presence of leukopenia; (5) the liability of copious gastro-intestinal hemorrhage from time to time; and (6) prolonged course without any tendency to spontaneous cure. Splenectomy is usually curative.

Rolliston, in discussing the relation between Banti's disease and splenic anemia, says: "The title Banti's Disease is now often used as synonymous with splenic anemia even by those who fully recognize that it is a sequel or terminal stage of splenic anemia, and does not occur in all cases even when unduly prolonged."

Rollins claims that Banti describes three stages of the disease named for him: (1) the preascitic stage in which splenic enlargement is present with or without anemia; (2) the transitional stage, of which the most prominent symptom is diarrhoea and at which time anemia and blood changes are found, the liver is somewhat enlarged and jaundice may be present; (3) the ascitic stage, or Banti's disease proper.

The normal function of the spleen is considered to be chiefly that of a mechanical filter, which removes from the blood degenerated red blood cells and toxic agents above colloid size, such as microorganisms and debris, on which it acts before sending them to the liver for further detoxication and elaboration. It also develops lymphocytes.

The spleen, when chronic splenic anemia develops, acts as a focus of infection, being a storehouse for infection whose toxic materials are dumped too rapidly into the liver, causing a cirrhosis to take place. It also undergoes changes of function which cause an overdestruction of red blood cells.

The relation of the spleen and liver in action has been compared to the glomeruli and tubuli of the kidneys, for the spleen extracts certain products which it passes on to the liver for disposal. This is true not only of waste products, but also of certain substances which the liver must return to the body to maintain normal life. With regard to the latter function, the spleen may be considered as a "scrapping plant," where the worn-out blood cells are delivered to have removed from them whatever may be of further service to the body.

Gato, in animal experiments, found that the amount of pigment in the bile was relatively diminished after splenectomy, both with and without the administration of hemolytic agents.

The spleen assists in producing immunity. This is indicated by experiments in which Morris and Bullock have demonstrated that splenectomized rats were more susceptible to ordinary infections, and to sublethal injections of live bacteria, than were others on whom abdominal castration had been done for controls. It is well known that the spleen can filter parasites of disease from the blood, but it cannot destroy these organisms. Probably the most striking example of this is the accumulation of plasmodia of malaria, the ague-cake of the tropics. The same is true of the spirochaeta pallida. The enlargement of the spleen in typhoid fever and chronic infections is

due to the accumulation of bacteria and the reaction to infection.

The spleen apparently does not initiate the pathologic processes with which it is concerned, but acts as a secondary agent.

Thus the spleen in splenic anemia shows marked fibrosis, including thickening of the capsule and trabeculae and changes of a fibrotic nature in the reticula. Endophlebitis is also sometimes to be found. The spleen pulp itself is greatly increased in amount, chiefly due to increased number of venules. Wartkin believes that in many, if not in all, cases of splenic anemia the underlying condition is obstruction in the splenic and portal circulation most commonly due to thrombophlebitis. The action of the spleen normally may be likened to that of a filter, removing not only old red cells, but also bacteria from the circulating blood, and we must suppose the ordinary acute splenic tumor to be, in part at least, in connection with this function. If the splenomegaly is the result of the isolation of malarial organisms, or of tubercle bacilli or the spirochetae of syphilis or other organisms, then we have a specific disease and we regard the enlargement of the spleen as merely one of the body protective measures. There must be, however, many similar splenic reactions against various bacteria or toxins. If this be pronounced, of fairly long standing, and accompanied by anemia of marked grade, we group the cases as splenic anemia or Banti's disease. This, at least, is the present status of the matter. This theory practically infers that the anemia is a secondary matter, and that the splenic enlargement is the primary thing. We could, of course, refer the anemia to hemorrhages, except for the fact that they are not constant and, in fact, are absent in some cases, showing very low red counts. The red cells of splenic anemia do not show an increased fragility. W. J. Mayo leans to the opinion that a fibrotic spleen destroys more red cells and that, in the absence of any special stimulation of the bone marrow, anemia must result. He suggests, too, that the disease changes may bring about reductions of the pigment production and thus actually reduce the hemoglobin balance.

In an exhaustive study, Krumbhaar considers that all diseases of the blood in which anemia is produced depend on the dynamic standpoint of the constant interplay of the blood-forming and blood-destroying apparatus, termed the hemolytopoietic system, and the adjustment there spoken of as the hemolytopoietic balance. The question, considered in regard to the anemia of splenic anemia, is whether this condition is due to an excess of blood-destruction or to a paucity of blood-formation. In considering the cause of the anemia the chief factors are the bone marrow, spleen, liver, lymph nodes and reticuloendothelial apparatus. By the close inter-relation of this system and the taking over of the function of one by another in disease, Krumbhaar explained the rapidity with which the blood picture approaches the normal after splenectomy.

In the Bradshaw lecture for 1920, Sir Berkeley Moynihan draws attention to the fact that the spleen is not a specialized organ with one or two particularly developed functions, but is one of a group of organs more or less interdependent. Failure to recognize this is, perhaps, the chief reason why the splenic functions have for so long remained obscure. Moynihan's conception of the problem is more illuminating, in that he considers the spleen to be closely connected with and, in fact, a part of four important systems of the body.

(1) *The spleen as a part of the hematopoietic or blood-making system.* Formation of red and white cells takes place in the spleen only during embryonic and early post-natal life, as a rule. In infections and leukemia, however, the spleen may take part in the production of white blood cells. The relation of the spleen to the bone marrow has already been discussed.

(2) *The spleen as a part of the reticuloendothelial system.* We have already described the endothelial cells as lying loosely in the reticulum of the spleen pulp. The liver also has certain large phogocytic cells, the star cells of Kupffer which are endothelial in nature. They are known to proliferate after removal of the spleen, and they may take up iron pigments. Lymphatic glands also contain phogocytic endothelial cells, which multiply after splenectomy. The bone marrow, too, contains somewhat similar cells. Thus, these structures seem definitely related morphologically, and we can more easily understand why blood pigment is found in the liver, lymph glands, and bone marrow when hemolyzing poisons are administered. It has been found, in studying the immature bodies of cholera, that the spleen and long bone marrow contains the largest proportions of these substances. Various other experiments on animals indicate that the spleen is an important power in resisting infective processes.

(3) *The spleen as a member of the digestive system.* All the blood of the spleen passes into the liver, carrying hemoglobin within the bodies of the wandering cells. The spleen, therefore, stands in an important relation to the liver. It is known, of course, that synchronous with digestion there is a slow expansion of the spleen, but what bearing this change may have upon so-called digestive leucocytosis is not clear. It is stated that splenic extracts cause intestinal peristalsis. A further connection with the digestive system lies in the probability that the spleen takes part in purin metabolism.

(4) *The spleen as a part of the sympathicoendocrine system.* It is assumed that the spleen has a sympathetical endocrine function, but it is impossible to prove it.

In a detailed resume, Connors considers cases of splenomegaly which may be classified from diagnostic standpoint into acute and chronic. I shall discuss here only chronic forms, the most important of which, from a differential standpoint, are myelogenous leukemia, lymphatic leukemia, Hodgkin's disease, chronic malaria, hemolytic jaundice, polycythemia-vera, pernicious anemia, Gaucher's disease, Von Jaksch's disease, syphilis (hereditary and acquired), tuberculosis, kala-azar, chronic infections, Still's disease, purpura, hepatic cirrhosis, obstruction to the portal vein, Amyloid disease, and tumors.

The diagnosis of splenic anemia depends on the existence of splenomegaly, varying degrees of anemia of the secondary type, usually leukopenia and a relative lymphocytosis, and the frequent occurrence of gastric and intestinal hemorrhages. Because of the hemorrhages, the condition is often diagnosed as gastric or duodenal ulcer, the splenic enlargement, even

though considerable, being overlooked or disregarded. It should be emphasized that in cases of gastric or intestinal hemorrhage, splenic anemia must be eliminated as a cause. In the later stages of the disease (Banti's disease) the existence of a very large spleen and ascites, due to the associated hepatic cirrhosis, together with the foregoing findings, usually suffice to make the diagnosis. Sometimes it will be impossible to separate the condition from primary hepatic cirrhosis with splenomegaly; the existence of a large liver and a comparatively small spleen or an atrophic liver with rather a small spleen, especially if ascites is present, favors a diagnosis of hepatic cirrhosis. Occasionally syphilis presents such a picture. Norris, Symmers, and Shapiro believe that all cases of so-called Banti's disease are due to syphilis.

The pathological findings and clinical records of sixty-nine patients with splenic anemia on whom splenectomy was performed at the Mayo Clinic from November 14, 1905, to September 1, 1920, were reviewed by Chaney in great detail. The most important points in his findings are as follows:

1. A composite picture of the pathological findings in the spleen in splenic anemia was found to be one of generalized fibrosis. There were no findings in the splenic tissue which would enable the pathologist to make a positive diagnosis of splenic anemia, yet the abnormality was as characteristic of this disease as in other diseases producing splenomegaly.

2. The average weight of the spleens was found to be 1015 gm.

3. The average age of the patients with splenic anemia was 33 years, and the number of males about equal to the number of females. There was apparently no familial tendency.

4. Their most common complaints were mass in the left abdomen, gastric hemorrhage, and weakness.

5. While abdominal pain was rarely given as the chief complaint, the histories brought out the fact that thirty-two of the patients had attacks at some state of the disease. In many instances the pain was probably due to perisplenitis.

6. In sixty-nine patients the average erythrocyte count was 3,700,000 Hg., 53 per cent; leucocyte count, 4990; coagulation time and the fragility tests were normal and the Wassermann tests and the stool examinations were negative.

7. A comparison of the number of lymphocytes in the differential count showed that the average was within the limits of normal. A lymphocytosis did not seem to be a characteristic in this series.

8. In the study of the liver tissue in the cases of splenic anemia, thirty showed a definite cirrhosis, but in none was the liver entirely normal.

9. Twenty-four of the patients with cirrhosis had ascites.

10. Twenty-three and three-tenths per cent of the patients with cirrhotic livers died within forty days of the operation, while within the same length of time the death rate of the remaining patients was only 12.8 per cent.

Zaccarelli, in 1549, and Ferrerius, in 1711, have been accredited, by some authors, with removal of the spleen, but these cases have been discredited by others for lack of sufficient detailed records. The earliest authentic cases of splenectomy are those reported by Quittenbaum of Rostock in 1826, Kuchler of Dermstadt in 1855, and Spencer Wells of London in 1866, each reporting a case of splenectomy.

In each case the patient died a few hours after operation. Plan, in 1867, did the first successful splenectomy from which the patient recovered.

From Collier's table of twenty-nine cases of splenectomy reported from 1549 to 1881, we find a mortality rate of 72.4 per cent. Van Wert's thesis of 1897 records 274 cases with 104 deaths, or a mortality rate of 37.9 per cent.

Modern surgical methods and proper selection of cases have reduced the mortality in splenectomy from all causes. Giffen, in 1921, reported a series of seventy-three splenectomies for splenic anemia in which nine deaths are recorded, a mortality rate of 12.3 per cent.

To illustrate what has been accomplished by splenectomy in splenic anemia, I would like to cite three cases from our clinic which present the three types of cases described by Hollins.

During the last two and one-half years, six cases of splenic anemia have been encountered, illustrating the types of the disease, progress, and operative cures.

CASE REPORTS

Mr. E. W. Age 33. Superintendent Water Company. First observation on January 8, 1924.

Present Illness—Following a fall in December, 1919, patient was ill three days and complained of weakness. Following this he had a very severe hemorrhage, vomiting blood and also passing a considerable quantity by bowel. At this time he was confined to bed for fourteen days. X-ray diagnosis was negative. His second hemorrhage was in August, 1920, at which time he vomited blood and was in bed one week. In May, 1921, the third hemorrhage occurred. Fourth hemorrhage January 4, 1924, at which time the patient passed blood by bowel two days. Since the first hemorrhage, the patient has complained of weakness, but feels that this has not been progressive. No symptoms referable to ulcer. No genito-urinary, cardio-respiratory, or sensory symptoms.

Physical Examination—General Appearance: Sallow and anemic. Throat: Infection of tonsils, second degree. Teeth: Dental sepsis, second degree. Blood pressure, 110/80. Abdomen: Slightly tender in upper left quadrant. Liver: Palpable; not markedly nodular. Spleen: Enlarged eight to ten times.

Temperature, pulse and respiration, normal.

Laboratory Findings—January 8, 1924, Hg., 42 per cent; r. b. c. 2,800,000; w. b. c. 4300; color index .7 plus; number cells counted 200; polys 48 per cent; small lymphs 31; large lymphs 16.5; large monos 2; transitional 2.5; slight anisocytosis; slight poikilocytosis; moderate polychromatophilia; coagulation time 6 minutes; fragility .28 per cent to .30 per cent increase in resistance. Blood Wassermann negative. Urinalysis negative.

On January 11, 1924, this patient was transfused with 450 cc. of blood, the indirect sodium citrate method being used, and six days later (Jan. 17, 1924) splenectomy was done. The following findings were made at operation: Stomach showed large vericosities over fundus and under pyloric end—no sign of ulceration of stomach. Liver showed little cirrhosis compared to average case of Banti's Disease. Gall-bladder was thick-walled, emptied easily and there were no stones. Spleen six to eight times normal size, with extensive adhesions over the surface, and was covered with thick plastic exudate of greyish color. The patient had an uneventful post-operative course and was dismissed on the fifteenth day post-operative in very good condition. By the first of March he felt very well and was allowed to resume light work. May 19, 1924, Hgb. 82 per cent, r. b. c. 5,140,000, condition excellent. Early in September, 1924,

the patient reported again for observation: hgb. 86 per cent; r. b. c. 4,650,000. Had resumed heavy work and felt himself entirely well. Very well at present time.

Mr. F. B. Laborer. Age 28.

Present Illness—Five years ago onset of present trouble began with a sudden attack of vomiting blood. Considered hemorrhage quite severe; was in the army and did not stop drill work. After this time felt fairly well until one year ago when he again had a severe hemorrhage from the stomach—this did not weaken him greatly. He felt normal after vomiting and continued to work. The third attack of vomiting was March 27, 1923; of the same nature and also passed blood by bowel; no marked weakness following this attack.

Physical Examination—Seems in good physical condition except for some pallor. Throat: tonsils atrophic. Abdomen: Spleen freely movable about 4 inches below costal margin. Liver enlarged about 4½ inches below costal margin. Blood pressure 165/90.

Laboratory Findings—March 29, 1924, Hgb. 45 per cent; r. b. c., 1,130,000; color index, 1.9 plus; w. b. c. 4400; 200 cells counted; small lymphs, 25.5; large lymphs, 2.5; large monos, 2; trans, 3; polys, 66.5; basophiles, .5; slight anisocytosis; moderate poikilocytosis; slight bassophilic stippling; moderate polychromatophilia; bleeding time 10 minutes. March 29, 1924, transfusion of 400 cc. of citrated blood was given. April 3, 1924, w. b. c. 3400; Hgb. 50 per cent; r. b. c. 3,470,000. April 16, 1924, splenectomy done; liver markedly cirrhotic; spleen five times enlarged, plastic lymph exudate over entire surface. Marked vericosities over stomach and diaphragm; no free fluid. Usual splenectomy done.

Reaction following splenectomy excellent. Convalescence was entirely uneventful. Within two months, the patient was working again, and has had no trouble. Recent Hgb. is 75 per cent; r. b. c., 4,500,000. Has had one hemorrhage since July 1, 1924, by bowel, moderately severe. Apparently not affected by hemorrhage and working again, as before.

Mr. E. L. M. Age 29. Examination, October 30, 1922. Complaint: Weakness and loss of appetite.

Present Illness—About 7 years ago began to lose weight and have a feeling of dragging and loss of ambition, with marked weakness on exertion. Would gain weight for a time and feel better; later would lose again. Has grown slowly and progressively worse with greater loss of strength. Night sweats for six months; no temperature or chills. Bleeding from gums and nose. No vomiting of blood or passing blood by bowel. Has noted enlargement of abdomen for last two years.

Physical Examination—Poor physical condition; sallow and emaciated. Teeth: pyorrhoea and dental sepsis III. Spleen very large, extending to umbillicus, and quite painful. Abdomen distended to a moderate degree.

Laboratory Findings—October 23, 1922. Hgb. 44 per cent; r. b. c., 3,860,000; w. b. c., 14,200, group III. On admission malarial parasites were demonstrated. The patient was given a course of intravenous salvarsan and intravenous quinine with one course of Deep Therapy over the spleen, over a period of six weeks. The night sweats disappeared and he felt much better, but the spleen grew larger. No transfusion given.

Splenectomy on October 4, 1922. Spleen densely adherent to diaphragm and lateral wall and about ten times normal size. Usual technique of operation. Considerable hemorrhage. Liver markedly cirrhotic. No gallstones. For six months following operation, patient had to be tapped five times, but finally began to get better and has gradually improved in strength. Has been working as foreman on a ranch for the last year. July 18, 1924, white count 5600; 31 small lymphs; 4 per cent large lymphs; 62 per cent transitionals; Hgb. 80 per cent; r. b. c., 4,130,000. Very well at present time.

Mrs. J. E. K. Age 29. October 10, 1922.

Present Complaint—Rapid heart and weakness for about four years, with palpitation of heart, excitability, tremor of hands following exertion—all symptoms mild in degree. Marked loss of strength. About three years ago began to have enlargement of upper abdomen which continued for six to seven months and gradually receded.

Physical Examination—Teeth, marked dental sepsis. Heart, moderate dilatation, systolic murmur over base and apex. Spleen enlarged three to four times. Considerable pallor.

Laboratory Examinations—Hgb., 23 per cent; r. b. c., 3,150,000; leucocytes, 4400; cells counted, 200; small lymphs, 23 per cent; large lymphs, 1.5 per cent; large monos, 2.5; trans, 4.0; polys, 68.0; eosin, .5; baso, .5; slight anisocytosis; moderate poikilocytosis; slight polychromatophilia. Urine examinations negative. The patient was given a transfusion of 500 cc. of citrated blood and advised to have all foci of infection removed as she had many bad teeth. Her tonsils were negative and there was no history of previous tonsillitis. Since this time the patient has improved very markedly in strength and activity. Has been kept on iron at intervals. Present Hgb., 72 per cent; r. b. c., 4,200,000; spleen and liver about comparable in size. Size of spleen has diminished until it is just palpable under the costal border. Blood picture about the same.

Miss H. S. Age 40. May 9, 1924.

Present Illness—January 11 began to have diffuse abdominal cramps and severe vomiting and chill; high white count 13,000; temperature 102. Operated elsewhere January 23, 1924; chronic appendix; liver and gall-bladder normal. Patient seemed to go into uremia afterward. April 2 had recovered sufficiently to be taken home. Hgb., 50 per cent. For two weeks stationary; some tendency to diarrhoea. April 28 some blood in vomitus; some acute pain in abdomen. On entry into hospital May 4, 1924, spleen was palpable 3 cm. below costal margin. Blood count: Hgb. 35 per cent; r. b. c., 2,410,000; slight poikilocytosis; moderate polychromatophilia; w. b. c., 6200; 200 cells counted; polys, 71.5; small lymphs, 21.5; large lymphs, 2.0; large monos, 2; trans, 2; eos, 1. Blood urea normal. P. S. T., normal. Transfusion of 500 cc. of citrated blood. Feces examination negative. Wassermann negative. Fragility increased. X-ray of chest negative. Since leaving the hospital has had a very severe hemorrhage with more transfusions. Was then diagnosed as toxic ulcer elsewhere. Has had two hemorrhages and two transfusions since leaving hospital. Blood shows same picture.

Miss H. G. Age 6. November 6, 1924.

Present Illness—April, 1923. At this time parents noted nose bleeds as often as four or five times in twenty-four hours. The child became languid and tired and did not wish to play with others, though she apparently had no particular weakness. Skin and whites of eyes, yellow.

The initial attack of jaundice lasted about two to three months. Since that time attacks of two to three days' duration every month or so. No pain at beginning of P. I., but shortly began to complain of severe colicky pain under umbilicus and the longer attacks of pain were usually associated with the jaundice mentioned. In addition many transient attacks of pain. Constant tenderness under umbilicus; occasional tenderness in pit of stomach. Vomited during early stages of this illness, with the attacks of pain and jaundice, but, during last eight months, has been nauseated with attacks without vomiting. Temperature ranging from 99½ to 101½, the latter being in August and September. Temperature rise usually in morning. Enlargement of the abdomen was first noted at the time of onset of the P. I.

Physical Examination—General Appearance: Child in good flesh and nutrition; seems somewhat anemic. Throat: Enlargement of tonsils with infection. Blood Pressure: Systolic 95; diastolic 50. Abdomen: When lying flat there is a definite enlargement of the upper left abdomen. On palpation spleen extends down to about 1½ inches above the umbilicus. Moves freely to about ½ in. of navel. Seems slightly tender to touch. Liver extends down about 1 to 1½ inches below costal margin. Slight tenderness below and around umbilicus. Temperature: Slight rise.

Laboratory Reports—Blood count; r. b. c. 3,450,000; Hgb. 65 per cent; color index .95; w. b. c., 3150; polys, 50 per cent; small monos, 49 per cent; large monos, 1 per cent. Blood examination: No parasites found. Slight anisocytosis, moderate poikilocytosis. Reticulated cells, 0.6 per cent. Myelocytes, none found. Urinalysis negative. Feces examination, negative. Urine examination for bile,

negative. Blood Wassermann, negative. Fragility test: Complete hemolysis, .32 per cent (NaCl); beginning hemolysis, .42 per cent (NaCl).

Diagnosis—Banti's disease (splenic anemia) chronic appendicitis. Immediate operation recommended, but, to date has not been performed. Child to return in March for splenectomy.

In considering this group of splenic anemias, it should be borne constantly in mind that the basic pathology of the spleen is generalized thrombophlebitis, resulting in secondary anemia, the initial onset coming as an acute thrombo-phlebitis, following most commonly influenza, or follicular tonsillitis. The probable bacteria is the streptococcus, which has a particular attraction for lymphoid tissue. Many spleens are found enlarged in influenza if a careful examination is made in severe cases. The fact that all individuals who have respiratory infections do not develop splenic anemia is due to resistance of the individual to streptococcus infections. The streptococcus, incysted in lymphoid tissue following an attack of follicular tonsillitis may result in rheumatism and endocarditis. Thus, when in an individual with lowered resistance the spleen is infected and repeatedly fed with more bacteria which it constantly strains out from the blood, fibrotic changes are initiated and the spleen itself becomes a focus of infection, the excess blood destruction probably being a toxic action from bacterial growth within the spleen. When these toxins, generated by the spleen, are poured into the liver, parenchyma cell destruction of the liver takes place and the destroyed parenchyma tissue is replaced by fibrous tissue. As the anemia becomes pronounced, the fibrosis and liver destruction increase until the ratio of liver destruction exceeds liver regeneration; then hepatic insufficiency and ascites occur. Remissions and exacerbations are caused by acute respiratory infections, bringing about a flare-up of thrombophlebitis.

The splenectomies reported are striking examples of this syndrome of development. In all, the respiratory or streptococcic etiology was the most probable. In one of the unoperated cases (Mrs. J. E. K.) there has been a marked improvement on one transfusion and removal of the focus of infection in the teeth. All have exacerbations of their anemia, with thrombo-phlebitis of the spleen following respiratory infection.

No case of splenic anemia should be splenectomized until careful and sufficient observations have been made. It is my belief that splenic anemia can be arrested in its early stages by removal of foci of infection, such as infected teeth and tonsils, which are the portals of entry of the streptococcus. The resistance of the individual at the same time should be strengthened by transfusions.

Splenic anemia in a well-advanced state should be splenectomized as soon as possible after careful preparation by transfusion and rest. If splenectomy is resorted to before an advanced degree of cirrhosis has taken place, complete and permanent cures are obtained. Great care should be observed not to operate in a state of acute thrombo-phlebitis with temperature.

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THE RELATIONSHIP OF THE CLINICAL PATHOLOGIST TO SURGICAL PRACTICE *

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The investigation of the end results of disease by Hunter and the physicians of his time gave impetus to the scientific investigation of disease. With the advance in the art of surgery there has been a parallel advance in the science of pathology, although in the early days of development the two were but remotely related. The extension of vision by the lens has cemented their union until the clinical pathologist has become indispensable to the progress of surgery. The necropsy pathologist will continue to correlate clinical, operative, and anatomic findings, seeking the truth, so that surgical deaths shall not have been in vain.

BY THE sense of sight and through the institution of methods of extending vision, it has become possible to explore still further the mysteries of disease, its cause and manifestations, and to base therapeutic measures on scientific principles.

The science of pathology and the art of surgery possessed little in common until the latter part of the nineteenth century. John Hunter definitely established the science of gross pathology, securing his specimens at necropsy, and made possible the teaching of pathology, not only from clinical subjects, but from actual material removed from the cadaver. Hunter's investigations revealed the ultimate result of disease and afforded opportunity for the study of effects of disease on the various organs of the body, in the gross and by the unaided eye. While the principles of magnification of vision by the microscope were evolved in the seventeenth century, before Hunter's time, yet magnification had not attained a power of more than 270 diameters and aided little in the examination of tissue. However, even so, the latter part of the seventeenth century disclosed the usefulness of the microscope in the cellular description of green plants, the malpighian corpuscles of the kidney, red blood cells, spermatozoa, and so forth. But not until the middle of the nineteenth century was knowledge of morbid anatomy based on the knowledge that the cell was the living unit. The description of the cell nucleus, the nucleolus, and protoplasm led to the cell theory of Schieden and Schwann and to Virchow's "cellular pathology," which was but the application of the cell theory of the botanist to human structures in disease.

The art of surgery, which is older than the science of pathology, owes its early advancement to the knowledge of gross anatomy and pathology, and to the later extension of vision by means of the microscope and various instruments for visual examination of the eye, larynx, esophagus, urinary tract and rectum, and by means of the roentgen ray.

Progress of surgery was retarded in the early days by the suffering occasioned by operative measures, and operations were resorted to only in dire necessity. However, with the discovery of anesthetics, surgery received a new impetus, and the access to the abdomen, chest, and head thus afforded, revealed diseased tissue within these cavities in life that hitherto had been seen only at post mortem. While

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